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RESISTANCE OF WESTERN WHITE PINE TO BLISTER RUST: A FOUNDATION FOR INTEGRATED CONTROL[1]

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ABSTRACT

Low-level resistance to blister rust in alliance with other control options such as hazard mapping, Ribes *eradication, rapid early growth of white pine, and branch-canker pruning can greatly expand the outlook for management of white pine in the West.*

KEYWORDS: *Cronartium ribicola; Pinus monticola;* computer simulation; epidemiology

White pine blister rust (*Cronartium ribicola*) has been a major forest disease in North America since its introduction at the turn of the century. We all remember control methods that were tried and discarded. Eventually, management of western white pine (*Pinus monticola*) on National Forest lands was suspended in 1968 (Ketcham and others 1968) until such time as resistant planting stock became available. Some resistant stock is currently available from Regions 1 and 6 of the USDA Forest Service. Outplantings of resistant materials have performed very well (Bingham and others 1973; Steinhoff 1971).

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High level resistance remains the best single solution, but much could be done to complement resistance and increase management flexibility through the application of our total arsenal of disease management techniques. Particularly promising is the use of low-level resistance, which can be quickly obtained from naturally selected seed (Hoff and others 1976) or extensive breeding programs. Some of the possibilities that can extend the usefulness of low-level resistance are hazard determination, *Ribes* eradication, branch-canker pruning, and accelerated early growth.

First, let us consider *low-level resistance alone.* Several kinds of resistance are known to exist in populations of western white pine (table 1). The effect of immunity imparting mechanisms is obvious, but, what do forms of low-level resistance mean in terms of stand damage? We have constructed a computer simulator (McDonald and others, manuscript in preparation) that allows us to evaluate various alternatives. Output from the simulator is used in the following discussion to demonstrate some possibilities. Input variables have been selected to simulate infection over a 100-year period at Pierce, Idaho.

Table 1.--*Observed resistance mechanisms in* Pinus monticola:Cronartium ribicola *system*

Mechanism of resistance	Resistance type	Hypothesized inheritance	h^2
1. Resistance in secondary needles to a yellow-spot forming race	Vertical	Recessive gene	
2. Resistance in secondary needles to a red-spot forming race	Vertical	Dominant	
3. Resistance in secondary needles to a yellow-green-island spot forming race	Vertical	Dominant gene?	
4. Resistance in secondary needles to a red-green-island spot forming race	Vertical	Dominant gene?	
5. Resistance in secondary needles that prevents spot formation	Vertical	?	
6. Reduced frequency of secondary needle infections	Horizontal	Nondominant gene?	
7. Slow fungus growth in secondary needles	Horizontal	Polygenic?	
8. Premature shedding infected secondary needles	Vertical	Recessive gene	
9. Fungicidal reaction in short shoot	Vertical	Recessive gene	
10. Fungicidal reaction in stem	Vertical	Oligogenic?	0.367
11. Slow fungus growth in stem	Horizontal	Polygenic?	0.21-0.46
12. Tolerance to infection	Horizontal	?	

In the first example we will assume a population of white pine where all the trees have the reduced needle-lesion trait. Mortality is expected to reach about 13 percent in 100 years, not 75 percent as would be the case for trees with no resistance. What would happen if growth rate of the rust in secondary needles could be slowed down so that one more year would be required for the fungus to reach wood, or if needle retention could be reduced to 2 years instead of 3, or if 3-year-old needles could be induced to drop after an unusually heavy infection year (Griffing and Ursic 1977)? The epidemiological result of all these situations is that the mortality curve would reach about 30 percent in 100 years.

We have evidence of a resistance mechanism that slows the growth of the rust on branches and boles. If growth rate were halved, then one could expect about 42 percent mortality in 100 years. Additional kinds of resistance are known and others may be discovered, but for the time being let us take a look at the combined effects of those just discussed. If all the trees contained all three of the mechanisms (reduced needle-lesions, slow fungus growth, and 2-year needle retention), then one could expect about 1 percent mortality from blister rust in 100 years. Although we should work toward this solution, difficulties could be encountered in trying to breed a population where each tree contained all three kinds of resistance. Additionally, we may wish not to put all our eggs in one basket. The rust and climate will surely vary, so we must retain as much genetic flexibility in our pine population as is possible. For this reason and others we should use only as much resistance as is necessary so that the largest gene pool possible can be maintained. In order to maintain a large gene pool, many disease management techniques in addition to resistance must be utilized.

Solution two is *hazard determination*. In my opinion, two *Ribes* species cause the bulk of the damage. These are the so-called upland species, *Ribes sanguinium* and *R. viscosissimum*. I believe this because in most situations basidisopores will not infect pine at a distance greater than about 1,000 ft. In addition, more than 90 percent of the spore load produced by an individual bush will not travel further than about 40 ft (Buchanan and Kimmey 1938). This does not mean that infection never occurs more than 1,000 ft from a bush. It most certainly does (Lloyd 1959; Van Arsdel 1972), but I do not think that such occurrences are in the majority. It is my belief that most trees that become infected will be within 50 ft of a bush. Furthermore, most infections on most trees are nonlethal branch cankers; therefore, damage, measured in terms of mortality rather than percent infected, should be directly related to the relative dispersal patterns of *Ribes* bushes and the white pine trees on most sites. *Ribes* species such as *R. bracteosum* and *R. petiolare* that are known for their ability to produce prodigious spore loads are restricted almost entirely to stream banks. Consequently, damage caused by this source should, in most cases, be local. The upland species, on the other hand, tend to be evenly spaced over the landscape.

The only direct evidence I have concerning this idea is from some experimental field tests that included susceptible materials (table 2). In the cases where upland *Ribes* were present and evenly dispersed, early mortality has been very high. When few upland bushes were present, little infection has resulted to date. These results are based on visual estimates of *Ribes* populations and counts of infected and dead trees. It is essential that dispersal patterns of both trees and bushes be determined on given sites and associated infection levels ascertained. In the absence of such data, we can look at simulation results. The simulator shows an obvious relationship between *R. viscosissimum* density and infection: Less than 1 percent dead after 100 years at one bush/ha; about 2 percent dead at 10 bushes/ha; about 80 percent dead after 100 years at 100 bushes/ha; and 100 percent dead after 45 years at 1,000 bushes/ha.

The reason for abandoning *Ribes* eradication was excessive long distance spread, but may I remind you that western white pine is still doing very well in northern Idaho. At this time it is difficult to say how much of the relatively low level of infection is due to natural selection of resistance and how much to lack of long distance spread.

Table 2.--*Determinations of blister rust hazard to susceptible white pine as a function of density of upland* Ribes

Area	Planting date	Infection measured		Upland *Ribes* density
		Year	Percent	
Skull Creek	1971	1975	>75	High
Priest River - middle elevation	1971	1977	>80	High
Ida Creek	1971	1977	< 5	Low
West Fork Merry Creek	1969	1976	>90	High
Gletty Creek	1969	1977	< 1	Low
Jaype	1970	1976	< 1	Low

The situation is not as simple as it appears. If we can assess hazard by some means of *Ribes* distribution mapping and site description, then I believe large areas of land would become available for planting easily obtained low-level resistant white pine. There is also the possibility that *Ribes* eradication could be used to advantage before planting or just after a regeneration cut. It could also be used to reduce damage to an acceptable level in certain stands already naturally regenerated. The time has come to take a new look at *Ribes* eradication.

How can one manage stands of low-level resistance to minimize losses? The third solution is to couple breeding for rapid growth with fertilization and other cultural techniques to *obtain rapid growth so that trees will pass through their most vulnerable stage quickly.* Trees are most vulnerable to damage during their early years while they are small. For example, if a stand growing on site class 60 was given one severe inoculation at 10 years of age, then 37 percent of the trees would be dead by age 35 years. If the stand was given the same inoculum load at age 25 years, 3 percent would be dead at age 50 years. This prediction by the simulator is based on the relationship of mortality to size, not age related variation in resistance. Also fast-growing trees apparently can "outgrow" the rust, which means a reduced level of mortality over a longer time span. Trees growing on a site with an index of 90 and given the inoculum load at age 25 are expected to show about 1.5 percent mortality by age 60, rather than 3 percent mortality at age 50 years as on a site indexed at 60. The assumption was made, in the absence of data to the contrary, that fungus growth rate is independent of tree growth rate.

If unexpectedly high levels of lethal infection are obtained such as would occur from a heavy inoculum year when a stand was young, then the fourth solution, *branch canker pruning*, is a possiblity. The major deterrent to this possibility is bole cankers resulting from infection of needles located on the bole. Fortunately, such infections are rare except at the highest infection levels because of the relatively small target such needles present. At 1,000 *R. viscosissimum* bushes/ha from age 15 to 20, about 150 trees/ha (original stand 2,000/ha) would have direct stem cankers. At 100 bushes (*R. viscosissimum*)/ha, less than 10 trees would have stem cankers. Since low-level resistance stock will be planted only on sites characterized by low *Ribes* populations, high densities of bushes (above 100/ha) should not be reached. Consequently, pruning of branch cankers should be a good backup solution in plantations and a reasonable alternative in some naturally regenerated stands, especially if used in conjunction with *Ribes* eradication.

There are still other combinations of resistance mechanisms, cultural treatments, biological controls, and chemical controls. The possibilities range from computer-assisted hazard prognosis to the incorporation of a biological control agent such as *Tuberculina maxima* (Wicker 1970), and the encouragement of natural inactivation of cankers (Hungerford 1977). An excellent opportunity exists for the development of an effective integrated control strategy that directs all management options toward a common goal--healthy and fast-growing western white pine.

PUBLICATIONS CITED

Bingham, R. T., R. J. Hoff, and G. 1. McDonald.
1973. Breeding blister rust resistant western white pine. VI. First results from field testing of resistant planting stock. USDA For. Serv. Res. Note INT-179, 11 p. Intermt. For. and Range Exp. Stn., Ogden, Utah.

Buchanan, T. S., and J. W. Kimmey.
1938. Initial tests of the distance of spread to and intensity of infection on *Pinus monticola* by *Cronartium ribicola* from *Ribes lacustre* and *R. viscosissimum*. J. Agric. Res. 56:9-30.

Griffing, C. G., and S. J. Ursic.
1977. Ethephon advances loblolly pine needle cast. For. Sci. 23:351-354.

Hoff, R. J., G. 1. McDonald, and R. T. Bingham.
1976. Mass selection for blister rust resistance: a method for natural regeneration of western white pine. USDA For. Serv. Res. Note INT-202, 11 p. Intermt. For. and Range Exp. Stn., Ogden, Utah.

Hungerford, R. D.
1977. Natural inactivation of blister rust cankers on western white pine. For. Sci. 23:343-350.

Ketcham, D. E., C. A. Wellner, and S. S. Evans, Jr.
1968. Western white pine management programs realigned on Northern Rocky Mountain National Forests. J. For. 66:329-332.

Lloyd, M. G.
1959. Air currents send spores over ridge. West. Conserv. J. 1959:32-33.

Steinhoff, R. J.
1971. Field levels of infection of progenies of western white pines selected for blister rust resistance. USDA For. Serv. Res. Note INT-146, 4 p. Intermt. For. and Range Exp. Stn., Ogden, Utah.

Van Arsdel, E. P.
1972. Environment in relation to white pine blister rust infection. *In* Biology of rust resistance in forest trees, p. 479-493. USDA For. Serv. Misc. Publ. 1221, 681 p. Washington, D. C.

Wicker, E. F.
1970. Retention of infectivity and pathogenicity by *Tuberculina maxima* in culture. Mycologia 62:1209-1211.

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